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A REVIEW OF CORN STUNT DISEASE (ACHAPARRAMIENTO) AND ITS INSECT VICTORS, WITH RÉSUMÉS OF OTHER VIRUS DISEASES OF MAIZE

Agricultural Research Service

UNITED STATES DEPARTMENT OF AGRICULTURE

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A REVIEW OF CORN STUNT DISEASE (ACHAPARRAMIENTO) AND ITS INSECT VECTORS, WITH RESUMES OF OTHER VIRUS DISEASES OF MAIZE

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Corn stunt disease, caused by a persistent virus, occurs in the United States in Arizona (36),[1] California (18), Mississippi (36), New Mexico,[2] and Texas (2, 25, 50). The same disease called "achaparramiento" is also known in many Central and South American countries. It is reported from Brazil,[3] Colombia (62), Costa Rica (19, 62), El Salvador (3, 4, 5, 6, 62), Guatemala (54, 55, 62), Honduras (62), Mexico (13, 34, 53), Nicaragua (32, 62), Panama (62), and Surinam (76).

The disease has caused irregular, severe, localized losses, but it is usually seen widespread in low or trace percentages of infection. Because of this, corn stunt has been considered a disease of economic importance only in the New World tropics and subtropics, with only scattered occasional losses in the Southwestern United States. This seemingly restricted distribution and minor importance of the disease in the United States was based on observations made over 20 or more years, when the known insect vectors of the virus were apparently limited to a relatively restricted plant host range in areas of very moderate climate.

A condition, appearing to be corn stunt, occurred in Mississippi in 1962 causing severe losses in Yazoo County. Maramorosch (36) confirmed that this condition in Mississippi was corn stunt disease by recovering corn stunt virus from field specimens collected late in 1962.

In 1963 this same condition reoccurred in greater severity in Mississippi and was noted in Louisiana. There was also a severe, extensive occurrence of what appeared to be corn stunt in southern Ohio. Late-season surveys in 1963 disclosed what may be foci of infection of corn stunt disease in Alabama, Georgia, Indiana, Kentucky, Missouri, Ohio, and South Carolina.

1/ Numbers in parentheses refer to Literature Cited at the end of this report.

2/ Personal communication with P. C. Duffield, Department of Agricultural Services, New Mexico State University of Agriculture, Engineering and Science, University Park.

3/ Personal communication with A. S. Costa, Instituto Agronomica, Secao de Virolgia, Campinas, Brazil.

This occurrence of what may be corn stunt east and north of the known range of the disease into the border of the Corn Belt could be serious. At least it indicates the need to review and perhaps to revise our considerations of this disease problem in the United States.

REVIEW OF CORN STUNT LITERATURE

In March of 1945 Frazier (18) reported an abnormal condition of corn that had been recurrent in Tulare and Fresno Counties in California for 3 years. He described the symptoms, indicating the damage caused to both field and sweet corn varieties, and observed a close association between the condition and the presence of the leafhopper Dalbulus maidis (DeL. & W.) (Baldulus). Frazier postulated that the condition could be due to a toxicogenic feeding effect of the insect or to a virus being transmitted by this species. Either possibility existed, since he could find no obvious relationship between the severity of the symptoms, the percentage of the plants affected, and the density of the leafhopper population. He also pointed out that plantings 100 percent affected with the condition had often been observed, and that early planted corn seemed to escape the condition.

Altstatt (2) reported a somewhat similar condition in corn in the Rio Grande Valley of Texas in June of 1945. A rather complete symptom description was given, which would seem to be a composite derived from observing the condition in sweet corn, popcorn, and field corn varieties. This description closely paralleled that given by Frazier for the condition in California. Altstatt stated that sweet corn and popcorn were more commonly affected than field corn, and he reported one field of Ioana sweet corn with a 30-percent loss of marketable ears. He also observed that early plantings suffered least from the condition.

Pickett et al. (50) in February 1946 reported the condition in corn in the Rio Grande Valley and described "symptoms and destructiveness" in greater detail. The symptoms were similar to those caused by "some viruses and insects," and the condition was compared with that caused by other viruses known to infect corn. These authors also indicated that the condition was probably not seed transmitted.

In 1946 Kunkel (30) reported that a virus had easily been transmitted from field specimens of corn in California and Texas by the leafhopper D. maidis, and he established that the condition was due to a virus infection and that maidis was an efficient vector of the virus. Since the symptom syndrome and the internal anatomy of infected plants differed sufficiently from those of other corn viruses, he thought that the causative agent was a hitherto undescribed virus. He therefore proposed the name "stunt" from one of the principal symptoms to avoid confusion with the name "streak" already applied by Storey and McClean (72) to a distinct virus transmitted by the leafhopper Cicadulina mbila Naude in Africa.

Kunkel (29, 30) in 1946 also established the fact that stunt virus required a long "incubation" period in D. maidis. In 1948 Kunkel (31) published the results of more extensive studies of stunt, giving the syndrome of the disease under controlled conditions, the biology of the vector D. maidis and vector-virus-host relationships. Negative evidence was presented on transmission of stunt virus mechanically, through the seed, and by the corn planthopper (Peregrinus maidis (Ashm.)), the vector of corn stripe, and the six spotted leafhopper (Macrosteles fascifrons (Stål)).

Kunkel (31) indicated that because of the long incubation period in the known hosts-corn and teosinte-stunt is typically a disease of plants that are approaching maturity. He also pointed out that spread of the disease to other parts of the United States was an open question depending on whether D. maidis could migrate north. Correlations were made between various vectors and viruses, including stunt virus, and the incubation periods in host plants. It was suggested that such correlations might depend on rates of virus multiplication in the vectors and therefore support the theory that plant viruses multiply in leafhopper vectors.

In 1950 Niederhauser and Cervantes (45) reported Dalbulus elimatus (Ball) (Baldulus) from Mexico as a vector of corn stunt virus with a slightly longer latent period than D. maidis. When they used D. elimatus as a vector of stunt virus, earliest symptoms in the host were seen 4 to 7 weeks after inoculation. They pointed out that the abundance of D. elimatus in the Mesa Central indicated that this species was probably responsible for much of the spread of stunt in this part of Mexico.

The biology, ecology, and distribution of D. elimatus and maidis in central Mexico were presented in detail by Barnes (9) in 1954. His principal findings relating to the corn stunt problem in the United States are as follows: D. maidis occurs most commonly below an altitude of 750 meters in tropical to semitropical areas and does not occur above approximately 2,000 meters in more temperate areas. D. elimatus on the contrary is most common above 750 meters. Barnes clearly distinguished between host plants-those on which the life cycle of the insects could be completed-and food plants-those on which the insects could maintain themselves as adults.

Maramorosch (34) in 1955 reported two distinct types of corn stunt in Mexico and separated them on a symptom basis. He designated the causative agents as Rio Grande and Mesa Central strains of stunt virus, because they were first found and described from these areas. He described and illustrated the differences between the two strains, indicating Mesa Central type virus infection in corn produced a more continuous diffuse striping and chlorotic spotting was less conspicuous or absent. Discoloration at the base of newly developed leaves was usually lacking in plants infected with Mesa Central virus, but the reddening was often very pronounced, with a deep purple commonly appearing in older leaves. The affected plants were more stunted than those with Rio Grande virus infection. Additional shoots were produced in the leaf

axils of plants infected with the Mesa Central strain, but such multiple shoots occurred mainly at the base of the plant. The symptoms of both strains of stunt virus were observed in Mexico in many corn varieties.

In 1958 Maramorosch (35) presented results from studies of cross protection between the Rio Grande and Mesa Central strains of corn stunt in the vectors D. maidis and elimatus. He confirmed the strain relationship, and both viruses were formally described.

Maramorosch (36) in 1963 reported finding the leafhopper D. maidis in the field on corn in Arizona in December 1962. He indicated that the presence of adults and several instars of this species so late in the season suggests a permanent rather than a transient population. Corn stunt virus was also recovered with known noninfective D. maidis from field plants collected near Phoenix, Ariz. Maramorosch is thought to be the first to report corn stunt disease in Arizona.

Corn stunt was also reported by Maramorosch (36) in 1963 as occurring in Yazoo County, Miss., in 1962. This report was based on recovery of the virus from field specimens collected in that county. He indicated that symptoms in the test plants were generally characteristic of those produced by corn stunt virus infections, but that they were not typical of the Rio Grande strain of the virus. Stunting and production of axillary shoots were evident in the test plants, but chlorotic streaks were rarely seen. The symptoms observed in the recovery from Arizona sources were also considered milder than those produced by the Rio Grande strain, but similar to those seen in the test plants infected from Mississippi isolations. The milder symptoms were observed in the greenhouse, but in the field the disease might be more severe.

Observations made during the 1963 outbreak of corn stunt in Mississippi proved it can be severe in the field.

SYMPTOMS OF CORN STUNT DISEASE

Composite descriptions of corn stunt are given here to aid investigators unfamiliar with the disease. These syndromes are derived from personal observation, discussions with investigators familiar with the disease, information from correspondence, and a review of the symptoms given in the literature. The descriptions are generalized, and it should be emphasized that not all the symptoms given will necessarily occur in all infected plants observed in the field.

The symptoms developed in corn infected with stunt virus depend somewhat on the age of the plant at the time of inoculation and the strain, or strains, of the virus involved.

"Early" Stunt Infection

Corn seedlings infected with stunt virus soon after emergence are most severely affected. The first symptom of the disease, seen 4 to 6 weeks after inoculation, may be small chlorotic spots of various sizes in the laminae at the base of the leaves developed after inoculation. These spots become more numerous and tend to coalesce into stripes along or between the veins of succeeding leaves. This striping may remain discrete or become slightly diffuse in succeeding leaves until only a general chlorosis is seen in the apical leaves in the cone of the plant. At this time the appearance of the plant may closely simulate that induced by certain nutritional disorders, such as subacute deficiencies of zinc or manganese. Purple to red coloration may develop in the leaves from the ends and margins inward and in the chlorotic areas as the stunting becomes evident, but there is no necrosis of the tissues. Concurrent with the development of these leaf symptoms, the elongation of the plant is slowed because of reduced length of the younger internodes, and the plants may almost cease growth. (See fig. 1.)

It is from this characteristic symptom that the name "stunt" was derived for the disease. Early infected plants may be severely affected and greatly dwarfed. They remain small and may produce no tassels or very weak ones. Ears may or may not be formed, but if they are, seed set will be poor or the ears may be completely sterile.

"Intermediate" Stunt Infection

The syndrome of stunt in plants infected during the second quarter of their growth will be slightly different from that of plants inoculated earlier. Chlorotic spotting and striping in the younger leaves may not occur. Should this be the case, the first symptom will be the development of a general chlorosis of the whorl leaves when the plant is about half grown. Elongation of the plant is slowed and the whorl leaves remain bunched. (See fig. 2.) The apical part of the plant at this time may superficially resemble the form of a feather duster.

As the disease progresses, the top part of the stalks is stunted because of the increasing shortness of the emerging internodes. The tassel will generally emerge smaller than normal with a scraggly appearance. The anthers may be shriveled, producing little or no pollen. Proliferation of basal or axillary shoots or ears is not usual in intermediate infections of stunt. The plants may produce an ear or two at nodes developed before infection, but seed set may be poor or the ears may be nubbins. (See fig. 3.) Frequently these plants will be sterile.

A purple or reddish coloration may appear in the older leaves after the onset of the initial whorl chlorosis. The development of the coloration may eventually involve all the leaves and the stalk of a plant. The coloration symptom may be followed by a drying of the margins and tips of the leaves so that the infected plants seem to mature sooner than the healthy plants in the same field. The onset of this coloration gives the diseased plants the appearance of chronic phosphorus deficiency.

"Late" Stunt Infection

Corn plants infected with stunt virus after they are half grown will generally grow to maturity without developing very definitive symptoms of the disease. Yet there may be a little stunting of the top of the stalk because of a slight shortening in the internodes developed after infection, plus varying degrees of development of a purplish to reddish coloration in the leaves, with an apparent hastening in the maturity of these plants compared to healthy plants in the same field. Ears of these plants may be normal or only slightly reduced in size. The overall appearance of these plants after they are half to full grown may closely resemble that of corn with late acute phosphorus deficiency.

Root Symptoms

Kunkel (31) described and illustrated root symptoms of corn stunt disease in the late stages. The virus is believed to move slowly into the roots, stunting them and causing development of numerous fine, many-branched rootlets, so that the root mass appears denser and more compact than normal. Kunkel indicated that the root symptoms are particularly striking when the brace roots are also affected. Symptoms were commonly seen on both brace and feeder roots in the affected areas in Mississippi and Louisiana. Color illustrations of corn stunt symptoms are given by Ullstrup (75) and Stoner and Ullstrup (69).

TAXONOMY OF CORN STUNT VIRUS VECTORS

The two leafhoppers Dalbulus maidis (DeL. & W.) and Dalbulus elimatus (Ball) are the only known vectors of corn stunt virus. D. elimatus was first described by Ball (8) in the genus Deltocephalus in 1900. D. maidis was first described and placed in the genus Cicadula by Wolcott (83) in 1923.

Oman (46) in 1934 erected a new genus Baldulus. He described the type species, Baldulus montanus Oman, and included Deltocephalus elimatus Ball in the genus. He did this because he considered the species to be congeneric with the type in spite of the peculiar, elongate genitalia of both sexes.

Oman (48) in 1949 published a comprehensive classification of the nearctic leafhoppers occurring in America north of Mexico. In this classification the genus Baldulus was included in the tribe Macrostelini, and the species Cicadula maidis DeL. & W. was moved to the genus Baldulus to become Baldulus maidis (DeL. & W.). The genus Baldulus then in 1949 included three species-montanus, elimatus, and maidis.

DeLong (14) in 1950, in view of increasing knowledge about biological relationships and the implication of species of Baldulus in the transmission of corn stunt virus, reviewed the taxonomy of this group in North America including Mexico. DeLong's study resulted in the erection of a new genus Dalbulus represented by elimatus (Ball), maidis (DeL. & W.), and four new species-guevarai, longulus, gelbus, and acus, which were described then. DeLong used Deltocephalus elimatus Ball as the type for the new genus Dalbulus. The genus Baldulus was then left with the type montanus and two new species-bilineatus and tropicus, which were described then. Both of these new Baldulus species are known only from Mexico.4/

DISTRIBUTION OF CORN STUNT VIRUS VECTORS

Dalbulus maidis

Dalbulus maidis was originally described (83) in 1923 from specimens taken at San Sebastian, Puerto Rico, and at Haina, Santo Domingo. Oman (47) in 1948 published the known distribution of this insect as follows: United States - Arizona (Patagonia, Sedona, Tucson); California (Alhambra, Colton, Temple); Florida (Sanford); North Carolina (Raleigh); and Texas (McAllen, Nueces County, Richland Springs). Foreign Countries - Argentina (Tucuman); Brazil (Campinas, Viscora); Costa Rica (San Pedro de Montes de Oca); Cuba; Dominican Republic; Mexico (Monoclava); Peru (Canete); Puerto Rico; and Venezuela (El Valle).

Barnes (9) in 1954 published extensive information on the distribution of D. maidis in central Mexico in the area bounded by latitude 18°5' N., the Pacific Ocean, latitude 22°5' N., and the Gulf of Mexico. Detailed collection data are given for 1950 through 1952. The species is found in this area commonly under tropical and subtropical conditions below an altitude of 750 meters and not above approximately 2,000 meters.

4/ J. P. Kramer briefly described D. maidis and elimatus and made a key for their determination and separation from some of the more common similar species. See U.S. Agr. Res. Serv., Plant Pest Control Div., Coop. Econ. Insect Rpt. 14: 105-106, Feb. 1964.

Dalbulus elimatus

Dalbulus elimatus was first described (8) in 1900 from three specimens taken at Santa Fe, Mexico. DeLong (14) in 1950 listed other locations of collection in Mexico as follows: La Guarda, Mexico City, Rio Frio, San Jacinto, Tlalpan, and Xochimilco, D.F.; Guadalajara, State of Jalisco; Sitacuara, Uruapan, and Zacapu, State of Michoacan; Puebla, Santa Rosa, and Tehuacan, State of Puebla.

Barnes (9) in 1954 also published extensive information on the distribution of D. elimatus in the central Mexico area indicated in the preceding section for D. maidis. Extensive collection data are given for D. elimatus from 1950 through 1952. This species is found most commonly at altitudes above 750 meters, where more temperate climatic conditions prevail.

All reports on the distribution of D. elimatus have been from Mexico. Corn stunt has been reported from areas where neither D. elimatus nor maidis is known to occur. This may constitute circumstantial evidence that these species or closely related ones are in these areas. In view of the more northerly temperate location of the suspected corn stunt outbreak in the United States in 1963, it would seem logical to suspect the presence of D. elimatus as a possible vector even though there are no records of its occurrence north of Mexico. This reasoning is based primarily on the fact that D. elimatus can live under more temperate conditions than maidis. However, the possibility of the presence of an unknown vector must not be ruled out, and all leafhoppers should be tested, particularly species related to the known vectors encountered on corn in the areas of suspected corn stunt infection.

HOST PLANTS OF VECTORS OF CORN STUNT VIRUS

Dalbulus maidis

Kunkel (31) reported that corn (Zea mays L.)5/ and teosinte (Euchlaena mexicana Schrad.) are the only host plants on which Dalbulus maidis could complete the life cycle. The insect was able to live for about 3 weeks on oats (Avena sativa L.), barley (Hordeum vulgare L.), sugarcane (Saccharum officinarum L.), rye (Secale cereale L.), sorghum (Sorghum vulgare Pers.), and wheat (Triticum aestivum L.). Oviposition occurred in both sugarcane and sorghum. The eggs hatched, but the insects did not reach the adult stage on either plant. The grasses Tripsacum dactyloides L., T. floridanum Porter ex Vasey, Polytoca macrophylla Benthem, and sudangrass (Sorghum vulgare var.

5/ Nomenclature is from the cited literature, or The Standard Cyclopedia of Horticulture (7) when only common names are used in the reference.

sudanese (Piper) Hitch.) were tested as possible hosts of D. maidis. Kunkel found sudangrass to be the third best [food] host of the insect after corn and teosinte.

Adults were able to live on the dicotyledonous plants carrot (Daucus Carota [sic] L.) and parsley (Petroselinum hortense Hoffm.) for about 2 weeks, but sugarbeet (Beta vulgaris L. var. crassa Alef.) and pot marigold (Calendula officinalis L.) were better food hosts, with the insects living on them for about 30 days. Oviposition occurred in pot marigold and the eggs hatched, but the nymphs were only able to live a few days on this host. However, nymphs that hatched on pot marigold lived to maturity when transferred to corn.

D. maidis has also been taken in the field on potato (Solanum tuberosum L.) by H. R. Yust at Ibarra and Pomasqui, Ecuador, in 1953.6/

Dalbulus elimatus

Barnes (9) in 1954 made extensive tests of possible host plants of Dalbulus elimatus. In these investigations clear distinctions were made between host plants, those on which the insect could complete its life cycle; food plants, those on which the adults could feed and survive for a considerable time; and plants where the leafhoppers would be considered as "casual visitors."

Corn and teosinte were found to be the best host plants for D. elimatus. The insects could also complete the life cycle on wheat and barley. Mortality of the nymphs was greater on wheat and barley than on corn and teosinte and greater on barley than on wheat. The investigations proved that wheat is a natural host and could serve as an alternate host in the absence of corn and teosinte.

Various wild and cultivated forage crops were tested by Barnes (9) as possible hosts of D. elimatus, since these plants are in the field in Mexico during the dry season when neither corn nor the small grains are available in the fall and spring. Oviposition and development of D. elimatus to the adult stage occurred on Bromus laciniatus Beal. and B. inermis Leyss in greenhouse and field tests. Even though Barnes considered these two grasses of minor importance as host plants, they could serve as hosts during critical periods and possibly be a point of major control efforts when population levels are low.

6/ Personal communication with Sr. Jaime R. Diaz Moreno, INIAP, Estacion Experimental, Santa Catalina, Ecuador.

Other plant species were tested by Barnes in which D. elimatus females oviposited and the eggs hatched, but the nymphs failed to develop to adults. These plants were Chloris virgata Schwartz, C. submutica H. B. K., Eragrostis silveana Swallen, and Phalaris sp.

It is assumed that the following species were suitable as food plants for D. elimatus: Aristida sp., Bouteloua curtipendula (Mich.) Terr., B. gracilis (H. B. K.) Lag., Cynodon dactylon (L.) Pers., Erianthus sp., Sorghum halepense (L.) Brot., S. vulgare, and S. vulgare var. sudanense.

Various broadleaved cultivated plants were tested by Barnes as possible hosts of D. elimatus in the greenhouse and insectary. No oviposition occurred in these plants and adult insects lived on them only 2 or 3 days. For this reason he designated the following plants as poor food plants and not hosts of D. elimatus: Crotolaria sp., Daucus Carota [sic], Medicago hispida Gaertn., M. sativa L., Melilotus officinalis (L.) Lam., Phaseolus vulgaris L., Solanum tuberosum L., Vicia Faba [sic] L., and V. sativa L.

D. elimatus then has six host plants: Corn, teosinte, wheat, barley, and two grasses-Bromus inermis and B. laciniatus, whereas D. maidis is only known to complete its life cycle on corn and teosinte.

HOST PLANTS OF CORN STUNT VIRUS

Tests Made With Dalbulus maidis

Kunkel (31) in 1948 made critical host range studies of corn stunt virus using Dalbulus maidis as the vector and virus sources originally transferred from field plants collected in Texas. Both monocotyledonous and dicotyledonous species were tested. Susceptibility to the virus in these investigations was based primarily on the expression of symptoms in the test plants. Virus recovery tests, however, were made from sudangrass and two varieties of sugarcane, with the possibility that these species might be symptomless carriers of the virus.

All plants tested proved negative as hosts of the virus except corn and teosinte. Species tested with negative results in Kunkel's studies are as follows: Monocotyledonous plants: Oats, jobs-tears (Coix Lacryma-Jobi [sic] L.), barley, Polytoca macrophylla, sugarcane, rye, sorghum, sudangrass, gamagrass (Tripsacum dactyloides), T. floridanum, and wheat. Dicotyledonous plants: Sugarbeet, pot marigold, carrot, and parsley.

Tests Made With Dalbulus elimatus

Cervantes et al. (13) in 1958 studied the resistance of various Mexican corn varieties to corn stunt virus with Dalbulus elimatus as the vector. Mexican virus sources of the Mesa Central strain were used in this study. Teosinte and various Tripsacum species were included because of their relationship to corn in the plant tribe Maydeae and because they are wild in Mexico. Other monocotyledonous and dicotyledonous plants included in the investigations correspond to some tested by Kunkel using D. maidis, and others were recorded by Barnes as food plants of both vectors.

Again in this study only corn and teosinte proved susceptible to the virus. However these investigations showed a difference in susceptibility in various local selections of teosinte; the cornlike Chalco strain was highly susceptible and a selection from Honduras was apparently resistant. Plant species testing negative to corn stunt virus infection in these investigations are as follows: Monocotyledonous plants: Avena sativa, Bromus inermis, Cyonodon dactylon, Eragrostis sp., Hordeum vulgare, Saccharum officinarum, Secale cereale, Sorghum vulgare, S. vulgare var. sudanense, Tripsacum lanceolatum Rupr., Tripsacum sp., and Triticum aestivum. Dicotyledonous plants: Medicago sativa, Melilotus albus Desr. "var." annua, M. officinalis, and Vicia sativa.

U.S. DISTRIBUTION OF KNOWN AND SUSPECTED CORN STUNT VIRUS INFECTIONS

Prior to 1963 corn stunt disease was not known to occur east of Texas or north of the southwest border States of California, New Mexico, and Texas. In 1963 Maramorosch (36) reported recovery of the virus from diseased field plants collected in Arizona and Mississippi in 1962. There was also a suspected area of corn stunt infection in Louisiana in 1962. Apparently the suspected areas of infection spread farther east and north. Figure 4 shows the known and suspected areas of infection of corn stunt disease in the United States by counties as reported and compiled from the literature at the end of 1963. Precise locations of known or suspected corn stunt infections are given below. Where locations are taken from publications, the appropriate citations are given. Many of the listed reports have not been published previously and were obtained by correspondence with the indicated individuals.

Positive Corn Stunt Locations

Arizona: Maricopa,[7/] Phoenix (36)
California: Fresno, Fresno; Tulare, Tulare (18)
Mississippi: Yazoo, Yazoo City (36)

7/ Underlined words indicate counties followed by cities in county.

New Mexico: Bernalillo, Albuquerque; Dona Ana; Valencia; P. C. Duffield, New Mexico State University, University Park

Texas: Bee, Beeville; Brazos, Bryan College Station; Cameron, Blue Town, Weslaco; Fayette, La Grange; Hidalgo, Donna; Lavaca, Yoakum; Nueces, Robstown; Willacy, Raymondville, Santa Rosa (2, 25, 50)

Suspected Corn Stunt Locations

Alabama: Autauga, Prattville; Marion, Hamilton; Morgan, Hartselle; Pickens, near Ethelsville; Sumter, Livingston; R. T. Gudauskas, Auburn University, Auburn, Ala.; J. W. Broyles (deceased), former pathologist, Agricultural Research Service, Starkville, Miss.

Georgia: Cook, LaConte; Tift, Tifton; Worth, Poulan; H. C. Cox, Southern Grain Insects Research Laboratory, Agricultural Research Service, Tifton, Ga.; W. J. Wiser, Tifton

Indiana: Dearborn; Jefferson; Switzerland; A. J. Ullstrup, Agricultural Research Service, Purdue University, Lafayette, Ind.

Kentucky: Bracken, east of Cincinnati, Ohio; Greenup, South Shore; Lewis, east of Cincinnati; Mason, east of Cincinnati; R. E. Hampton, University of Kentucky, Lexington; Warren N. Stoner, Northern Grain Insects Research Laboratory, Agricultural Research Service, Brookings, S. Dak.

Louisiana: Avoyelles, Bunkie; Bossier, Curtis; Caddo, Caspiana; East Carroll, Grimes; Natchitoches, Natchez; Ouachita, Monroe; Rapides, Alexandria, Lecompte; Tensas, Avondale, Somerset; Thomas P. Pirone, Department of Plant Pathology, Louisiana State University, Baton Rouge.

Mississippi: Alcorn; Atalla, Kosciuska; Benton; Chickasaw; Choctaw, near Ackerman; Clay, West Point; Hinds, Edwards; Holmes, Durant, Goodman, between Durant and West; Itawamba; Leake, Carthage, Ofahoma, Wiggins; Lee; Leflore, Greenwood; Lowndes, near Columbus, Madison, Millville, between Tunetta and Millville, Tunetta; Marshall; Monroe, Amory; Montgomery, Kilmichael; Newton; Noxubee, Northeastern; Oktibbeha, Longview, Starkville, Sturgis; Pontotoc; Prentiss; Rankin; Scott; Sharkey, Anguilla; Sunflower, Parchman; Tippah; Union; Warren; Washington, Greenville, Stoneville; Wayne; Webster, Mathiston; Winston, Louisville; Yazoo, Yazoo City, all the county; W. A. Douglas, J. W. Broyles (deceased), G. K. Parris, all at Mississippi State University, State College; Warren N. Stoner, Northern Grain Insects Research Laboratory, Agricultural Research Service, Brookings, S. Dak.

Missouri: New Madrid, Malden; Oregon; Stoddard, Sikeston; A. J. Keaster, University of Missouri Delta Center, Portageville; M. L. Fairchild, Department of Entomology, College of Agriculture, University of Missouri, Colombia

Ohio: Adams, Blue Creek; Brown; Clermont, New Richmond; Gallia; Hamilton, southeastern Ohio; Highland; Hocking; Jackson; Lawrence; Meigs, Portland; Pickaway; Pike; Ross, Chillicothe; Scioto, Lucasville, Portsmouth; Vinton, McArthur; Washington; W. R. Findley, Agricultural Research Service, E. J. Dollinger, Department of Agronomy, L. E. Williams, Department of Botany and Plant Pathology, all at Ohio Agriculture Experiment Station, Wooster; B. F. Janson, Extension Division, Ohio University, Columbus, Max E. Maichele, Ortho Division, California Chemical Company, Toledo; Warren N. Stoner, Northern Grain Insects Research Laboratory, Agricultural Research Service, Brookings, S. Dak.

South Carolina: Richland, Columbia; Graydon Kingsland, Department of Botany and Bacteriology, Clemson College, Clemson, S.C.

Negative Reports on Corn Stunt Occurrence

Correspondence with many individuals concerning the distribution of corn stunt disease has made it possible to delimit areas where it is not known to occur. In certain instances such inquiry was made of individuals in locations adjacent to known areas of infection in an attempt to define the broad limit of the range of the disease. Negative evidence is not conclusive. However, investigators reported "not known to occur," "no record of occurrence," or simply "does not occur" for many areas.

These reports are listed here as a possible aid to those who may be interested in conducting surveys for the disease.

Arkansas: Negative, 1963; J. L. Dale, Department of Plant Pathology, University of Arkansas, Fayetteville

Colorado: Negative, 1963; L. E. Dickens, Cooperative State University, Fort Collins, Colo.

Connecticut: Negative, 1963; Saul Rich, Agricultural Experiment Station, New Haven, Conn.

Delaware: Negative, 1963; J. W. Heuberger, Department of Plant Pathology, University of Delaware, Newark

Florida: Negative, 1963; H. C. Cox, Southern Grain Insects Research Laboratory, Agricultural Research Service, Tifton, Ga.; G. F. Weber, College of Agriculture, University of Florida, Gainesville

Illinois: Negative, 1963; M. C. Shurtleff, A. L. Hooker, and H. H. Thornberry, University of Illinois, Urbana

Iowa: Negative, 1963; Don Peters, Department of Entomology, and R. C. Lambe, Cooperative Extension Service, Iowa State University, Ames

Kansas: Negative, 1963; W. H. Sill, Jr., Department of Plant Pathology, Kansas State University, Manhattan

Maryland: Negative, 1963; O. D. Morgan, Department of Plant Pathology, University of Maryland, College Park

Massachusetts: Negative, 1963; C. J. Gilgut, Department of Plant Pathology, University of Massachusetts, Amherst

Michigan: Negative, 1963; Donald Cation, Department of Botany and Plant Pathology, Michigan State University, East Lansing

Minnesota: Negative, 1963; Thor Kommedahl, Institute of Agriculture, University of Minnesota, St. Paul

Montana: Negative, 1963; E. R. Hehn, Department of Plant and Soil Science, Agricultural Experiment Station, Montana State College, Bozeman

Nebraska: Negative, 1963; J. M. Daly, Deparment of Plant Pathology, University of Nebraska, Lincoln

Nevada: Negative, 1963; S. D. Lyda, Department of Plant Science, Agricultural Experiment Station, University of Nevada, Reno

New Hampshire: Negative, 1963; Avery E. Rich, Department of Botany, University of New Hampshire, Durham

New Jersey: Negative, 1963; J. L. Peterson, Department of Plant Pathology, Rutgers University, New Brunswick, N.J.

New York: Negative, 1963; C. W. Boothroyd, Department of Plant Pathology, Cornell University, Ithaca, N.Y.

North Carolina: Negative, 1963; D. E. Ellis, North Carolina State College, School of Agriculture, Raleigh

North Dakota: Negative, 1963; R. G. Timian, Plant Pathology Department, State University Station, Fargo, N. Dak.

Oregon: Negative, 1963; R. A. Young, Oregon State University, Corvallis

Pennsylvania: Negative, 1963; J. S. Boyle, Department of Plant Pathology, 204 Buckhout Laboratory, Pennsylvania State University, University Park

South Dakota: Negative, 1963; Warren N. Stoner, Northern Grain Insects Research Laboratory, Agricultural Research Service, Brookings, S. Dak.

Tennessee: Negative, 1963; J. O. Andes, Department of Plant Pathology, Agricultural Experiment Station, University of Tennessee, Knoxville

Utah: Negative, 1963; O. S. Cannon, Department of Botany and Plant Pathology, Utah State University, Logan

Virginia: Negative, 1963; C. W. Roane, Department of Plant Pathology, Virginia Polytechnic Institute, Blacksburg

Washington: Negative, 1963; G. W. Bruehl, Department of Plant Pathology, Washington State University, Pullman

Wisconsin: Negative, 1963; G. S. Pound, Department of Plant Pathology, College of Agriculture, University of Wisconsin, Madison

Wyoming: Negative, 1963; G. H. Bridgmon, Department of Plant Pathology and Horticulture, University of Wyoming, Laramie

Negative Reports on Corn Stunt in Canada

Information given here was obtained by personal communication with the noted individuals. D. W. Creelman, editor, Canadian Plant Disease Survey, reported "Our records of the Canadian Plant Disease Survey, which go back to 1921, do not record the incidence of corn stunt on either field corn or sweet corn in this country since that time. The only virus disease recorded on corn is one occurrence of ring spot in southern Ontario."

Manitoba: Negative, 1963; John Giesbrecht, Canada Department of Agriculture, Research Branch, Experimental Farm, Morden

Ontario: Negative, 1963; E. E. Gamble, Department of Crop Science, Ontario Agricultural College, Guelph; C. G. Mortimer, Canada Department of Agriculture, Research Branch, Research Station, Harrow

Quebec: Negative, 1963; W. E. Sackston, McDonald College of McGill University, Quebec

L. S. Donovan of the Canada Department of Agriculture at the Central Experimental Farm, Ottawa, also reported negatively on corn stunt and indicated that neither Dalbulus maidis nor elimatus is known to occur there.

POSSIBLE SOURCES OF RESISTANCE TO CORN STUNT VIRUS

Cervantes et al. (13) in 1958 presented information on the resistance and susceptibility of various races of maize to corn stunt virus infection in Mexico. Generally they found the so-called tripsacoid races of corn tend to be resistant and the nontripsacoid races to be susceptible. This grouping of the corns investigated was based on the work of Wellhausen et al. (80, 81) dealing with the races of corn in Mexico. The investigations of resistance were conducted mainly in the Mesa Central region of Mexico, and there is some conjecture that the observed resistance may only be valid for the Mesa Central strain of corn stunt virus. Nevertheless, the races Zapalote Chico, Tuxpeño, Tepecintle, and Vandeño showed a high resistance under the test conditions (13, 53). These corns are characterized by Wellhausen et al. (78, 79, 80, 81) and Cervantes et al. (13).

Preliminary data of Cervantes et al. (13) also indicated a range of resistance to corn stunt virus in different varieties or types of teosinte.

Ancalmo (3) in 1961 reported on resistance tests in El Salvador with corn varieties from the Rockefeller Foundation, numbered Mexican varieties, and commercially available sweet corn. Comparative yields and percentage infectivities under infection with four "types" of corn stunt were used. These types were set up on the basis of different symptoms. None of the corn tested proved immune, but there was a high degree of resistance or tolerance in several varieties from the Rockefeller Foundation Mexican Program, particularly Poey T-23, T-46, T-62, and T-63. The variety Corneli 54 performed well in the comparative yield tests. Four lines resistant to stunt in Mexico resisted the virus in El Salvador as well. These were V-520-C-265 No., V-520-C-129-6, V-520-C-322, and V-520-C-128-14. Thirteen commercially available sweet corn varieties all proved to be highly susceptible. Investigations are being conducted at the Cotaxtla Experiment Station in Mexico to find sources of resistance to the Rio Grande strain of corn stunt virus.8/

Should the corn stunt area continue to enlarge and the disease be as severe as it was in 1962-63 in the United States, these investigations and the literature cited may become of particular interest to corn breeders.

OTHER CORN VIRUSES OCCURRING IN THE UNITED STATES

Several viruses are reported infecting corn in the United States. In some cases corn is considered the primary host; in others it is indicated in a host range where the virus in question is considered primarily in another

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8/ Personal communication with A. E. Rodriguez, CIB, Department of Plant Pathology, El Horno, Chapingo, Mexico

host. In these studies corn may be included as an experimental host or as being naturally infected. Most of these viruses are easily separable from corn stunt virus on the basis of symptomology, host range, physical-chemical properties, method of transmission, and vector relationships. Some of the viruses infecting corn are given here, with pertinent references should they be needed by investigators conducting surveys for corn virus diseases. There is no implication as to their relative importance in the order of listing.

Sugarcane Mosaic Virus

Brandes first described a mosaic disease of sugarcane and other grasses in 1919 (10). Corn was included in the host range of this study. In 1920 Brandes (11) described "corn mosaic," which was later proved by Brandes and Klaphaak (12) in 1923 to be caused by sugarcane mosaic virus infections.

The most conspicuous symptom of the disease in corn is an irregular, mottled, or mosaic appearance of the leaves. The pattern is usually more noticeable in the younger leaves and is caused by irregular development of interrupted streaks or lines in the long axis of the leaf. These streaks vary in size and may be confined between the veins or may irregularly cross the veins. Infected plants are generally lighter green than normal.

This virus is carried by aphids, particularly the corn leaf aphid (Rhopalosiphum maidis (Fitch)), and it is mechanically transmissible. Holmes (26) and Smith (63) listed corn in the host range of sugarcane mosaic virus. Ullstrup (75) included this virus in his publication on corn diseases in the United States. Sugarcane mosaic virus infections in corn are presumed to occur at least in the areas in the United States where susceptible sugarcane varieties are grown.

Cucumber Mosaic Virus

Corn has been susceptible to at least six strains of cucumber mosaic virus. Wellman (82) described southern celery mosaic virus infection in corn. Price (52) later showed this virus was a strain of cucumber mosaic. Pound and Walker (51) included corn in a host range of X1 cucumber mosaic virus as showing both local and systemic symptoms. In the same study a strain designated X2 was recovered from corn as a symptomless carrier.

The author in 1948-49 sucessfully inoculated corn with known sources of a western strain of cucumber mosaic virus, and recovered this virus from naturally infected field sweet corn plants. In the same experiment corn was experimentally inoculated with the Oklahoma and Bindweed strains of cucumber mosaic virus in separate series. Each was recovered individually after

systemic infection. Western, Southern, Oklahoma, and Bindweed strains of cucumber mosaic are lethal in systemic infections of corn seedlings.

The young center leaf wilts and turns a gray green, and the growing point deep in the whorl is killed. In older plants systemic infection results in the development of a broad yellow stripe frequently running the entire length of the leaf. Mesophyll formation may be completely suppressed so only the epidermal layers are present, forming a "window" in the striped area. Frequently the older leaves become purple as the disease progresses. In the final stages of the disease the tips of the whorl leaves may die and trap the growing point so the plant twists and hooks downward.2/

Various strains of cucumber mosaic virus are carried by several species of aphids. This virus is mechanically transmissible. Smith (63) listed corn in the host range of cucumber mosaic virus. Ullstrup (75) discussed the southern strain of cucumber mosaic in corn under "celery stripe." Infection of corn with cucumber mosaic virus is presumed to occur in the areas of known distribution of the various virus strains.

Maize Leaf Fleck Virus

Stoner (68) in 1952 described maize leaf fleck virus. Corn is its principal plant host.

The first indication of the disease is the development of small, roughly circular, pale spots in the interveinal area of the tip of the older leaves. These spots gradually enlarge to about 2 mm. in diameter, taking on a yellow hue. Then spots begin to appear toward the base of the leaf and may involve 1 to 2 inches of the length daily. When the terminal third of the leaf is spotted, the tip and margins begin to dry. The tip drying then follows the spotting toward the base of the leaf at about the same rate. A marginal burn or drying is very slow in developing and involves only a quarter inch on each side of the leaf. As the drying progresses the spots do not coalesce or in any way tend to produce streaking. After symptoms appear in the basal leaf, a similar development starts in the next alternate leaf above. Thus, there is a progression of the disease up the plant as well as a progression of symptoms in the individual leaves.

Leaf fleck virus is only known to be transmitted by the aphids Rhopalosiphum prunifolae (Fitch), R. maidis, and Myzus persicae (Sulz.). It is not mechanically transmissible. Leaf fleck disease is limited in known distribution to the San Francisco Bay area of California. Smith (63) also

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2/ Unpublished data.

considered it primarily a virus of corn. Ullstrup (75) included leaf fleck in his treatment of diseases of corn in the United States.

Corn Mosaic Virus

There is some ambiguity in the literature concerning corn mosaic. Kunkel (27) investigating transmission of a corn mosaic in Hawaii in 1922 proved that the corn planthopper (Peregrinus maidis) was the vector. He was able to transmit the Hawaiian virus from corn to corn but not from corn to sugarcane. In a later study Kunkel (28) reported the failure of P. maidis from North Carolina to transmit the virus of sugarcane mosaic of the United States to corn. Hadden (21) working on insect transmission of sugarcane virus in Hawaii found that two insects transmitted the virus. The corn leaf aphid (Rhopalosiphum maidis) transmitted a "mosaic" from various grasses to cane and and other grasses; the corn planthopper transmitted a "mosaic" from corn to corn. Sugarcane mosaic virus is now thought to have been involved in the aphid transmission studies by Hadden and corn stripe virus (see section on Maize or Corn Stripe Virus) in the transmissions by the corn planthopper. Therefore, what had been termed corn mosaic by Brandes was probably caused by infections with sugarcane mosaic virus, and the work of Kunkel involved the corn stripe virus.

Finley (16) in 1954 isolated a virus from naturally infected plants of Golden Cross Bantam sweet corn. No vector transmissions were attempted with this isolate. However, mechanical transmission studies indicated that the host range, symptom syndrome, and physical-chemical characteristics of this virus differed from those of sugarcane mosaic virus and cucumber mosaic virus.

Symptoms of corn mosaic are small broken chlorotic streaks on the younger leaves. These streaks then develop as intermittent or continuous interveinal streaks through the sheaths but not the stalks in systemic infections. Affected plants are dwarfed, and yield of marketable ears is reduced.

A mosaic disease of corn in the United States closely resembles that caused by sugarcane mosaic virus infection, but it is caused by a different distinct virus. The name "sweet corn mosaic virus" was used by Finley. Smith (63) listed this as "maize mosaic virus 2." This virus is also distinct from corn stripe virus (see section on Maize or Corn Stripe Virus).

Wheat Streak Mosaic Virus

Two viruses-Marmor virgatum var. typicum and M. virgatum var. viride-described by McKinney (37) are here considered together in relation to infection of corn. Host range studies of wheat streak mosaic virus by McKinney (39), Sill and Agusiobo (58), and Finley (17) include many varieties of field corn,

sweet corn, and popcorn. There is a range of susceptibility in corn from immunity to a fairly high susceptibility; the sweet corn variety Golden Giant is one of the most susceptible.

There is apparently a considerable range of symptoms caused by wheat streak mosaic virus in corn, with some indication that certain varieties may be symptomless carriers of the virus. Finley (17) gave the syndrome of the disease in the sweet corn inbred line P39 and in the hybrids Golden Giant and Golden Cross Bantam. McKinney (37) discussed symptoms in Golden Giant, and Sill and Agusiobo (58) found essentially the same reaction of Golden Giant as did McKinney.

Mechanical transmissions of the virus give the following generalized symptoms: First, diffuse local lesions may be irregularly scattered over the leaves. These lesions tend to run parallel along the veins but can cross over, giving a long, elliptical form in some cases. Systemic infection may or may not follow. Where systemic invasion occurs, a mosaic pattern develops, which resembles other "mosaics" of corn. A general chlorosis can develop on the tips of older leaves. General yellowing and stunting may or may not occur depending on the variety infected. All these symptoms can be and are frequently evanescent. Ears on infected plants may be subnormal and kenel set irregular.

The virus is mechanically transmissible and has been experimentally transmitted to corn by Sill and del Rosario (61) using the eriophyid mite vector Aceria tulipae (Keifer). It is presumed that corn could be found infected in areas where the virus occurs in the United States, particularly Kansas, Nebraska, South Dakota, and the Great Plains region into southern Alberta, Canada.

Barley Stripe Mosaic Virus

Corn has been given as an experimental host of barley stripe mosaic virus by McKinney (40, 41, 42) and Sill (57). The variety Golden Giant has specifically been used and gives the following reactions when mechanically inoculated. Seedlings are rarely killed, but in general the plants are definitely stunted. There may be some general yellowing. The disease is characterized in systemic infections by the development of long, broad, yellow or "bleached" stripes usually on the younger or whorl leaves. The stripes may be so numerous that the entire leaf may appear involved.

There is apparently no known insect vector, but the virus is known to be seed borne in several barley varieties. This virus is listed by Smith (63) as "barley false-stripe virus." Distribution includes Minnesota and South Dakota, but since the virus is seed borne it is presumed to have a much wider distribution.

Bromegrass Mosaic Virus

McKinney et al. (43) first indicated that bromegrass mosaic virus was distinct from the wheat viruses in 1942. Results of further studies by McKinney (38) published in 1944 included corn in the host range and indicated the general reactions to infection. The virus was described as a new species and characterized under the name "Marmor graminis" by McKinney (37) also in 1944. The corn variety Golden Giant is particularly mentioned and symptom descriptions are given in this variety. Sill and Pickett (60) reported severe damage to greenhouse grown corn by this virus in 1955. In 1956 McKinney (42) made some comparisons of bromegrass mosaic virus, barley stripe-mosaic virus, and wheat streak mosaic virus in Atsel barley as a test host. Symptoms of the virus were again given. In Golden Giant sweet corn seedlings many local lesions develop after mechanical inoculation with bromegrass mosaic virus. Systemic infection follows with necrosis, and the plants are ultimately killed.

Sill (57) in 1956 indicated the use of corn and Agrotricum (Agropyron X wheat) hybrids for a more rapid means of identifying the viruses of wheat streak mosaic, barley stripe mosaic, and bromegrass mosaic, indicating differentiation as follows: "On a seedling corn WSMV caused local lesions on a few varieties and diffuse systemic infection without stunting on others. Some were immune. BMV caused severe systemic infection, characterized by stunting and broad yellow to white stripes, usually on the younger leaves." This agrees with parts of McKinney's (37, 38) earlier work. Barley stripe mosaic virus can further be separated from the others since it is the only one of the three that is seed borne. Smith (63) listed Golden Giant sweet corn as a differential host for bromegrass mosaic virus.

There is no known insect vector for this virus. Bromegrass mosaic occurs in Kansas, but since the host range of the virus includes many widely grown grasses such as Kentucky Bluegrass (59), it is not unlikely that it is more widespread.

Barley Yellow Dwarf Virus

There are conflicting reports on the susceptibility of corn to barley yellow dwarf virus. Oswald and Houston (49) using the apple grain aphid (Rhopalosiphum fitchii (Sand.)) as a vector and a strain of the virus from California reported that the corn variety Golden Cross Bantam was immune. Watson and Mulligan (77) using the bird-cherry oat aphid (Rhopalosiphum padi L.) as the vector and a virulent strain of the virus from Kent were able to transmit the virus to the corn variety White Horse Tooth and induced slight stunting. They were also able to recover the virus from the affected plants. Allen (1) using the apple grain aphid as the vector and six strains of the virus induced symptoms in the variety Golden Cross Bantam with strain 11 of the virus from Arizona. He gave the symptoms as follows: "Interveinal

streaking and purpling that started at the tips of the oldest leaves and progressed downward on such leaves. As the plant matured, the symptoms appeared progressively higher on the plant, eventually becoming evident on the newer leaves." Allen's attempts to transfer the virus from the affected corn plants to other healthy corn, barley, and oat plants were unsuccessful.

Corn may be considered in the host range of barley yellow dwarf virus, but successful transmission seems to depend on a particular combination of the strain of the virus, the vector used in the transfer, and the corn variety selected as a test host. Since various strains of barley yellow dwarf virus are widespread and several aphid species are known to be vectors, it is possible natural infections of barley yellow dwarf virus in corn could be found in the field and reported.

CORN VIRUSES NOT KNOWN TO OCCUR IN THE CONTINENTAL UNITED STATES

Many virus diseases of corn that cause serious losses in other countries are not known to occur within the continental United States. In certain instances the insect vectors or closely related species and host plants of the causal viruses do occur here. The viruses could be inadvertently introduced, and if they became established, major problems in corn production could result. Most of these viruses can be differentiated from ones occurring here on the basis of symptoms, host range, physical-chemical properties, and vector relationships. Brief characterizations of some of these viruses and pertinent literature are given below.

Maize or Corn Stripe Virus

The word "stripe" has been used in descriptions of several diseases of corn, but it is perhaps most properly applied to the virus disease described by Stahl (67) in 1927 and listed by Smith (63) as maize mosaic virus 1 and by Holmes (26) as Fractilinea zeae (Holmes). The first symptom induced in corn by infection with maize or corn stripe virus is a few elongate chlorotic lesions at the base of the younger leaves. These lesions may fuse to form continuous stripes. Leaves emerging later from the whorl may be striped or banded in various patterns or both.

The virus is not mechanically transmissible, and is "persistent" in the vector the corn planthopper. Corn stripe virus has also been reported from Hawaii, Tanganyika, Mauritius, Trinidad, and Cuba. Maize stripe virus is also

common in Australia.10/ The disease called enanismo rayado, striped dwarf, described by Malaguti (33) in Venezuela, may be caused by this virus. The disease caused by this virus has also been called white stripe and Cuban white stripe disease.

Maize Streak Virus

Maize streak virus was reported by Storey (70, 71) and Storey and McClean (72) as causing "Streak disease of maize" or simply "streak." It is listed as "maize streak disease virus, Storey" by Smith (63) and as Fractilinea maidis (Holmes) McKinney by Holmes (26). The initial symptom of the disease is pale spots developing at the base of the first formed leaves. Younger leaves, developed after initial symptoms are apparent, may show chlorotic spotting and streaking. The later symptom is characteristic and gives rise to the name "streak" for the disease and the causal virus.

The virus is not mechanically transmissible, but it is persistent in its Deltocephaline leafhopper vectors Cicadulina bipunctella Matsumura, C. mbila, C. storeyi China, and C. zeae China. Streak disease is reported as a limiting factor in corn production in Natal, and the author has seen complete loss of crops due to streak infection in the western region of Nigeria. It has been noted in many parts of Africa as far north as Egypt.

Wallaby Ear Disease Virus

Wallaby ear disease of maize was first described and attributed to a virus infection by Schindler (56) in 1942. McKinney (37) in 1944 included the virus in his revision as a new species, Galla zeae. Holmes (26) also included the virus as Galla zeae McKinney, but Smith (63) listed it as "maize Wallaby-ear virus (Schindler)." The initial symptom of the disease is small swellings on the secondary veins on the ventral leaf surface. In young plants galls then develop rapidly along the veins basipetally. The plants are dwarfed and become abnormally dark green. Pollen yield is reduced, silk growth is slowed, and the cobs and kernels are underdeveloped.

The virus is not transmissible mechanically. The only known insect vector is the leafhopper Cicadula bimaculata Evans. This disease occurs in south-eastern Queensland, Australia. According to N. E. Grylls, "Severe symptoms on young maize seedlings often result in death of the plants. Some plants recover from a fairly severe infection and leaves showing gall formation slough off. Wallaby ear disease is present in the northern river area of New South Wales and Queensland every year." 10/

10/ Personal communication with N. E. Grylls, Division of Entomology, CSIRO, Canberra City, Australia.

Maize Rough Dwarf Virus

The disease caused by infection with maize rough dwarf virus was first reported from Italy by Fenaroli (15) in 1949. A detailed description of the disease was given by Grancini (20). Harpaz et al. (24) reported rough dwarf in Israel for the first time in 1958. In 1959 Harpaz (22) reported transmission of a pathogen from corn to corn by hypodermic injection of centrifuged sap extracted from plants showing typical symptoms of rough dwarf and established the viral nature of the disease. Insect transmission studies reported by Harpaz (23) in 1961 indicate that the areaopid Calligypona marginata (F.) = Delphacodes striatella Fallen is a vector of maize rough dwarf virus.

Symptoms of the disease will vary depending on the age of the plant at the time of infection, but the general syndrome is as follows: First the infected plant becomes darker than normal green. Small galls then develop on the veins of the undersurface of the leaves. The name of the disease is partially derived from the coarse, rough texture the galls impart. The brace roots may split and become necrotic. New roots are stunted. Leaf symptoms continue to develop as interveinal chlorotic streaks, which may coalesce to form large whitish areas. A reddish streaking may develop from the leaf margins and eventually involve the entire lamina. The plant's failure to elongate normally, because the length of the internodes is greatly reduced, accounts for the "dwarf" part of the name. This may be followed by degeneration of the apical bud if the leaves fail to open normally. Tassel formation may be completely suppressed or the developed tassels will be stunted and shed little pollen. Ears may be greatly shortened because of atrophy of the distal end of the ear and consequent failure of kernels to form. Rough dwarf disease occurs in Italy and Israel.

Oat Pseudo-Rosette Virus

Maize is included in the host range of oat pseudo-rosette virus reported from Russia (65). This virus has also been known as oat pupation disease virus, oat "zakuklivanie" (64, 66) disease virus, and Siberian oat mosaic virus. It was described by McKinney as Fractilinea avenae (37) and is so identified by Holmes (26). It is given by Smith (63) as "oat pseudo-rosette disease virus." Symptoms of the disease in corn are not specifically given.

The virus is transmitted most efficiently by the first- and second-instar nymphs of the leafhopper Delphax striatella Fallen. The fifth instar is believed to be immune, but the virus can overwinter in the insect (73). The virus is not mechanically transmissible nor is it transmitted through the seed or soil. The disease occurs over most of Siberia from the Pacific coast to the Ural Mountains, but especially near Omsk.

Sunflower mosaic virus is reported as mechanically transmissible by Muntanola (44) and Traversi (74). It is said to be transmitted by the onion thrips (Thrips tabaci Lind.), the green peach aphid (Myzus persicae), and the greenhouse whitefly (Trialeurodes vaporariorum (West.)). Maize is included in the experimental host range of this virus, but detailed symptoms of the disease are not given for corn. The virus occurs in Argentina and is presumed to be present in Russia, since it is carried in sunflower seed and was recovered from Russian seed stocks.

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Figure 1.-Field corn plant showing severe stunting caused by "early" infection with stunt virus, Mississippi, 1963.

Figure 2.-First symptoms of "intermediate" stunt virus infection of field corn, Mississippi, 1963. Note reduction in length of youngest internodes and consequent "bunching" of whorl leaves.

Figure 3.-Fully developed symptoms at maturity after "intermediate" stunt virus infection of field corn, Mississippi, 1963. Note the proliferation of many ears and poor seed set.

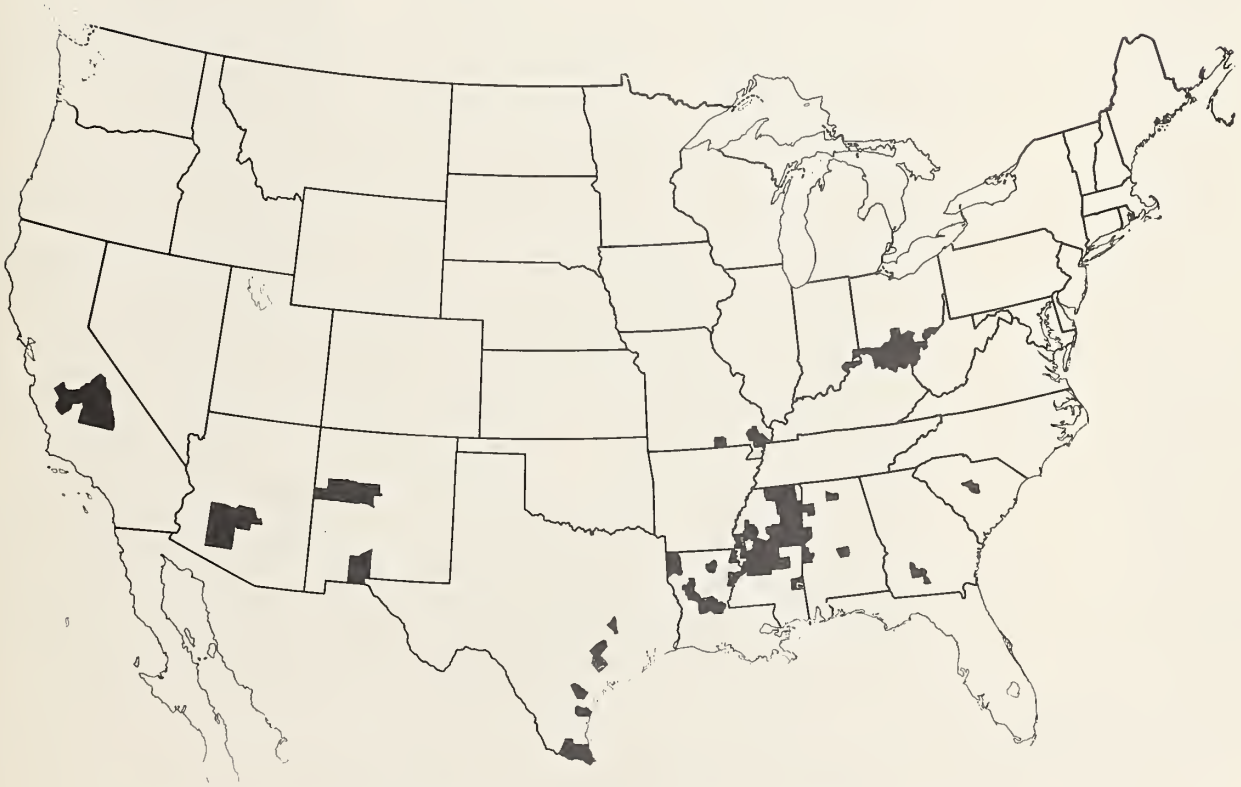

Figure 4.-Map of United States showing counties or parishes where corn stunt is known or suspected to occur, 1963.

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